your care complains of marked discomfort from light, tearing, pain in eye or brow, tenderness of globe, blurring of sight, or there be redness of the eye, note at once the location and character of the injection and the presence of secretion or not aside from tears. Carefully inspect the size, shape and activity of the pupil, also the color of the iris, then estimate the tension of the eye, taking his normal eye as a standard or your own if necessary.

The symptoms to be noted in iritis are ciliary injection, i. e., a purplish circumcorneal zone, a somewhat contracted, irregular and inactive pupil, discolored iris, tenderness over the ciliary region and practically normal tension; there will be hazy vision and pain complained of, especially at night; the patient is usually between the ages of 20 and 40 years. Promptly use atropine strong enough and often enough to produce and hold a wide, round pupil.

In glaucoma you have the ciliary injection, blurred vision, neuralgic pain, but you will have a dilated pupil and increased tension of the globe, with a patient over 40 years of age. Avoid atropine as here it is fatal to the eye.

In conjunctivitis you will get a redness more superficial and diffused than the ciliary injection, and it is not distinctly circumcorneal. There will be more or less secretion in the conjunctival sac or found on the lashes. The pupils are equal in size and activity. The pain is not severe—excepting in gonorrheal infection. One must not forget, however, that there is frequently a combined picture of conjunctivitis and iritis. In this case it is safe to act on the diagnosis of iritis, provided a glaucomatous condition has been eliminated. Exclude glaucoma by carefully noting the tension, next instill homatropine, an irregular pupillary effect will then be characteristic of iritis.

I have found rather a confusion of ideas in the minds of general physicians as to whether homatropine or atropine should be used—for diagnostic purposes use homatropine, for treatment atropine as a general thing is correct.

Particularly do not permit the absence of one or more of the classical symptoms enumerated in each ocular disease to disturb your diagnosis based on other unmistakable diagnostic signs.

The facts I particularly wish to call attention to are: the free and intimate connection of the vascular iris with the entire body through the circulation; the few and positive diagnostic signs for differentiating iritis, during disease or in emergencies open to all medical men by means of their unaided faculties; to the inestimable good that may come from the prompt use of a drop of atropine at the right time in iritis; to the tragedy of using that same drop of atropine in beginning glaucoma; to the not uncommon farce of a doctor prescribing that oft-abused drug, boric acid, for incipient iritis; and the value to the community of a conscientiously trained medical man in the fundamentals of ophthalmology.

AURICULAR FIBRILLATION.*

By H. W. ALLEN, M. D., San Francisco.

Of the numerous advances in our knowledge of cardiac pathology that have come as a result of the study of the heart by graphic methods, there is probably none of more importance to the physician than the recognition of auricular fibrillation as a clinical entity. Known among physiologists for some years as an experimental phenomenon it is only within the past three years that its occurrence among human beings has been definitely proven and as a result of this demonstration we now recognize it as the commonest form of persistent irregularity of the heart.

Very soon after the introduction of the sphygmograph into clinical medicine a type of pulse curve characterized by gross irregularity became the object of especial study. It received various names as pulsus irregularis; inequalis; deficiens; it was also termed the mitral pulse owning to its frequent occurrence in the late stages of mitral disease. Among other causes it was attributed to delirium of the heart. Somewhat later with the introduction of the polygraphic method of investigating cardiac disease this group of absolutely irregular hearts became the object of study by numerous workers and many new facts and much speculation were advanced concerning it. Mackenzie in particular devoted much time to the subject and contributed much to our present knowledge of the venous curves in this condition. He demonstrated the association of gross irregularity of the heart with systolic pulsation of the veins of the neck and gave to the latter condition its present name of the "ventricular form of venous pulse." Inasmuch as no sign of auricular activity could be detected in the venous curves, Mackenzie assumed that the auricle in these absolutely irregular hearts was paralyzed, a view that was agreed in by other observers. Later he was forced to alter his views because he found instances in which the irregularity of the heart ceased and normal action was restored. As this finding was inconsistent with the assumption of auricular paralysis he adopted the view of the nodal origin of the rhythm. This hypothesis explained very satisfactorily the absence of signs of auricular contraction in the venous curves inasmuch as Mackenzie assumed that auricle and ventricle contracted together in response to a common impulse generated in the auriculo-ventricular node of Tawara. It did not, however, satisfactorily explain the gross irregularity of the ventricle, nor did any of the other hypotheses that were advanced, such as great auricular asthenia or the occurrence of multiple extra systoles originating in the neighborhood of the sinus. In fact though this absolutely or per-

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petually irregular heart was carefully studied by many observers, it practically defied analysis until the advent of the electrocardiograph.

Certain writers, however, were on the right track. In 1907 Cushny and Edmunds reported a case of paroxysmal irregularity of the heart and drew attention to the similarity between the radial tracings from their patient and arterial curves from instances of auricular fibrillation seen experimentally in dogs. They suggested that a similar explanation might suffice for both. However, the first real demonstration of the unity of the absolutely irregular heart and auricular fibrillation came in June 1909, when Rothberger and Winterberg published a paper giving electrocardiographic curves from experimentally produced fibrillation in dogs and from patients with absolutely irregular hearts. They pointed out the exact correspondence of the two pictures but as they had had an opportunity of studying only two patients they contented themselves with suggesting that auricular fibrillation would be found to be the explanation of "some" cases of grossly irregular heart.

Later in the same year Thomas Lewis published an extensive article based on numerous animal experiments and electrocardiographic study of thirty human beings and proved very conclusively that auricular fibrillation is the fundamental condition in absolute irregularity of the heart. He has since amplified this proof and his findings have been fully confirmed by others, so that we may accept it to-day as fully demonstrated that the absolutely irregular heart associated with the ventricular form of venous pulse is due to the occurrence of auricular fibrillation. We have also come to regard this condition as a definite clinical entity and when present its name should replace or take precedence over the older designations of valvular disease and of myocarditis.

The proof that auricular fibrillation occurs in man and is the cause of the absolutely irregular heart is based on a careful comparison of the graphic records, polygraphic and electrocardiographic, obtained from patients suffering from this irregularity with similar records obtained from dogs or other animals in whom auricular fibrillation has been experimentally produced. The records are in complete accord.

The arterial curves in man are characterized, as has been stated, by gross irregularity. This picture is best seen when the pulse rate is rapid though it holds good at practically all times. There is an indiscriminate mixture of beats of all kinds; strong and weak beats and long and short pauses. There is no correspondence between the length of pause and the strength of the succeeding beat; a strong beat may follow a short pause or vice versa. As the pulse slows there is a greater tendency to regularity but except when complete dissociation of auricle and ventricle is present there is probably never absolute regularity.

The venous curves are characterized by the occurrence of the ventricular form of venous pulse, that is, the type in which all the prominent peaks fall consistently within the limits of ventricular systole. There is a complete absence of all evidence of normal auricular contraction. Occasionally during a long diastole of the ventricle when the vein is slowly filling there occur fine oscillations on the venous curve which can be referred to an incoordinate action of the auricle.

The electrocardiographic records are quite characteristic. A curve representing a normal cardiac cycle presents three main peaks or oscillations; first a small peak called P which is due to the contraction of the auricle; second, a tall peak called R which represents the beginning of systole; and third, a broader, more rounded peak called T corresponding to the end of systole. Other oscillations called Q and S are of minor importance and need not concern us. Between these phases the string of the galvanometer is at rest. The picture from patients with absolutely irregular hearts is quite different. There is no evidence of the normal P variation; no evidence of any coordinate auricular contraction. Instead we find numerous fine oscillations occurring throughout the cardiac cycle; the string of the galvanometer is never at rest. The ventricular peak R is present at irregular intervals throughout the curve; it is unmodified in appearance because the ventricular contraction wave has pursued its normal course and because R represents a very quick deviation of the string. The peak T, however, is variously modified, depending upon how these new oscillations have been superimposed upon it. The rate of these oscillations is variable: from 300 to 500 per minute. They are distinctive for they occur in no other cardiac condition so far known. It has been clearly shown that they are not due to any extraneous influences. By means of special leads from the chest wall Lewis has shown that they are maximal in the region of the right auricle and that when leads are chosen to show chiefly ventricular action they practically disappear from the picture.

Auricular fibrillation is easily produced experimentally in dogs by applying a faradic current to the auricle. Graphic records obtained during such an experiment are alike in all respects to those from patients with absolutely irregular hearts. The arterial curves exhibit the same gross irregularity; the venous curves show no sign of the normal auricular wave and finally the electrocardiographic records are exact duplicates. The conclusion is justified, then, that auricular fibrillation occurs in man and is the cause of the condition that we have hitherto known as absolute irregularity of the heart. It explains fully the otherwise puzzling features of the arterial and venous curves, the gross irregularity and the absence of the wave due to auricular contraction. For when fibrillation sets in there is a complete cessation of coordinate contraction; if viewed directly the auricle is seen to be in a position of diastole and at first glance it may appear to be absolutely at rest. If carefully inspected, however, its surface is seen to be the seat of great activity; constant undulations are everywhere present. The appearance is somewhat

similar to the very fine tremor sometimes observed in a protruded tongue or the fibrillary movements seen in skeletal muscles in some nervous disorders, only the activity is very much greater. In place of giving rise to a single rhythmical impulse which is conveyed to the ventricle, the auricle gives rise to exceedingly numerous irregularly spaced impulses which are conveyed along the auriculo-ventricular bundle without any semblance of rhythmicity and to which the ventricle responds as best it may. Hence the rise in rate and the gross irregularity of the pulse. So far as I know the human auricle has not actually been seen in fibrillation but the opportunity will undoubtedly occur sooner or later in the course of surgical work on the chest.

Regarding the pathological changes that must be responsible for the production of fibrillation, there is as yet no uniformity in reports. Inasmuch as the normal contraction of the heart is started in the sino-auricular node we might expect to find this structure extensively diseased and such in fact has been the case in some instances. Draper has recently reported a careful microscopical study of a heart that had shown fibrillation. He found a high degree of fibrosis in the sino-auricular node and extensive pericardial changes in its immediate neighborhood. In addition the walls of both auricle and ventricle showed varying grades of fibrosis. Other observers have reported somewhat similar findings though the node has not necessarily been extensively damaged. Most are agreed that the fibrotic and inflammatory changes in the walls of the right auricle constitute the essential pathological factor. Naturally extensive damage may be found in other parts of the heart in the way of valvular and myocardial disease and not infrequently the auriculo-ventricular bundle of His shows involvement, which fact can be utilized to explain the slow pulse that occurs in some cases of fibrillation.

Some of the clinical features of auricular fibrillation deserve mention. It is the most frequent irregularity of the heart with which we meet. Lewis has carefully analyzed a considerable group of persistent irregularities and the figures show that fibrillation makes up approximately 50% of the whole number. Of etiological factors the rheumatic infections head the list. It is convenient to divide fibrillation cases into two groups, the first, which may be called the endocarditic, embracing all those with rheumatic history or whose cardiac condition can be referred to a previous endocarditis, and the second, which may be called the sclerotic group, making up the remainder. When so divided it is found that about 70% fall in the first class. This grouping is of some help in estimating the probable response to treatment.

Of the symptomatology of the condition it is unnecessary to say much. Symptoms when present are mostly those of muscular insufficiency and do not require special mention. The effect of fibrillation on the murmur of mitral stenosis should be noted. The typical presystolic rumble disappears inasmuch as the auricle is no longer contracting and its place is taken by a murmur, usually of a rumbling quality, which occurs earlier in diastole. When the heart rate is rapid this murmur may occupy the whole of diastole, but when the rate is slow one can easily make out during the longer pauses that it occupies a mid-diastolic position and ceases entirely before the beginning of the first sound. That this is actually the case has been proven by graphically recording simultaneously the heart sounds and murmurs and the contraction wave by means of two string galvanometers.

Fibrillation when once established is usually a permanent condition. Patients showing this condition have been followed over periods of five and ten years and have always given evidence of its presence. It need not of necessity interfere with the heart's ability to withstand hard work. Patients are encountered with fibrillation who are able to perform hard labor without unusual discomfort. As in other cardiac disorders the ability of the heart to respond to demands upon it depends largely on the integrity of the ventricular muscle.

There is a not inconsiderable group of patients who exhibit a paroxysmal form of fibrillation, the attacks lasting for various periods from a few minutes to a number of days. In the intervals between attacks the pulse is regular and graphic records show that the auricle is contracting coordinately. During the course of severe acute infections fibrillation may be met with as a temporary phenomenon.

From what has been said of the importance of graphic records in this condition it might be inferred that they are essential for its recognition but such is not necessarily the case. Typical instances associated with decomposition are usually easily recognized; the combination of rapid rate with marked irregularity is almost distinctive. With pulse rates under 100 per minute confusion may occur with other forms of irregularity such as multiple extra systoles. If one increases the pulse rate by exercise the latter irregularities tend to disappear while those due to fibrillation are exaggerated. The final decision in doubtful cases must rest with the electrocardiogram.

Prognosis depends largely on two factors: our ability to maintain the heart beat at a moderate rate and the quality of the ventricular muscle. Fibrillation is itself an expression of disordered muscle but the damage may be quite unevenly distributed and the ventricular muscle relatively spared. The members of the endocarditic group of cases generally respond to treatment better than do those of the sclerotic group. The possibility of sudden death from fibrillation of the ventricles should be borne in mind.

In speaking of treatment I shall omit all mention of agents other than drugs and of these the only ones requiring discussion are digitalis and strophanthus. Where these fail others will not succeed. The results of treatment in auricular fibrillation are in general most satisfactory and it is from just these cases that digitalis has ac-

quired its great reputation as a cardiac drug. The chief effect of digitalis is seen in a reduction of the ventricular rate. This is brought about by the production of a partial block in the auriculo-ventricular bundle so that many of the auricular impulses fail to get through to the ventricle and the chamber is enabled therefore to perform more effective work. Digitalis produces this block probably in two ways: through its influence on the vagus and by direct action on the auriculo-ventricular bundle. The degree of reduction that can be brought about is sometimes remarkable; in fact complete dissociation of auricle and ventricle has been produced. In some cases only sufficient medication is necessary to reduce the pulse to normal limits, the heart then maintaining itself at the desired rate; in others varying doses of digitalis are required to keep the heart within bounds after the initial reduction; in still others, chiefly members of the sclerotic group, practically no lowering of the rate can be accomplished despite large doses. The outlook in these latter is naturally bad.

Our chief aim, then, in medication is to reduce the ventricular rate to normal limits and to maintain it at this level and we give sufficient digitalis to accomplish this purpose. No exact rules for dosage are therefore possible. If signs of digitalis intoxication appear, chiefly nausea and vomiting, we must, of course, stop its administration. Of the other possible effects of digitalis, the tonic action on the ventricular muscle, the diuresis, etc., it is not my purpose to speak; their influence is secondary to that of the reduction of rate.

Strophanthus in the form of strophanthin, given intravenously, has a definite place in the treatment of fibrillation. In patients seen for the first time with decompensation, rapid ventricular rate and great distress, much may be accomplished by a few timely doses of this drug. Its action is entirely similar to that of digitalis but results are accomplished much more quickly. In 8 or 10 hours one may attain a result that would require two or three days of digitalis by mouth to accomplish. The usual intravenous dose recommended, one milligram, is too large and may be the cause of sudden death. It is better to follow the plan advised of giving a small dose, such as ¼ milligram, and repeating in two or three hours for three or four doses. Agassiz has reported a series of cases treated by this method with very excellent results. The effects of a few doses given in this way are sometimes manifested for a number of days; subsequently digitalis by mouth may be resorted to as required.

In the treatment of paroxysmal fibrillation it is recommended that digitalis medication be not resorted to unless conditions are rather urgent. It is thought that this drug tends to prolong the paroxysm and in some instances to make it permanent.

THE FUTILITY OF CARDIAC STIMULANTS IN SHOCK.*

By SAXTON TEMPLE POPE, M. D., San Francisco.

With modern surgical methods and with trained anesthetists, we see less of surgical shock than formerly. But it seems to me that these highly trained anesthetists—because of their skill in avoiding dangerous phases of anesthesia, and consequently seldom coming in contact with shock—are particularly weak in their treatment of this condition. When any untoward condition arises during the course of surgical narcosis, and especially where there are cardiovascular phenomena connected with it, it is almost an invariable custom of anesthetists to resort to hypodermic injections of one or more of the various so-called cardiac stimulants.

Many of these drugs are not stimulants at all, but may best be classified as cardiovascular irritants. Some really inhibit the normal cardiac stimulation—such as digitalis, the action of which is mainly one of interrupting the conduction of stimuli from the sino-auricular node to the ventricle. The majority of these drugs are cardiac accelerators, and some have absolutely no legitimate excuse for being used at all.

The demonstrations of Crile, years ago, almost completely destroyed the popular confidence in the use of strychnine and nitroglycerine in shock. But, turning from one false support in their hour of trial, surgeons have placed their faith in equally futile or harmful drugs: camphor and caffein.

In the first place, all hypodermic medication is too slow in its action to meet the emergency of shock. It is an absolute waste of time to turn to the anesthetic table and to administer a hypodermic injection, when we know that under the most favorable circulatory conditions, a physiologic action of the drug cannot be expected within ten minutes. In the second place, we are putting dependence upon a measure which has no possible bearing on the disturbed function before us.

Shock, from a mechanical conception, is characterized by a rapid fall in vasomotor tension, and from a certain standpoint this is the one criterion of shock and is the one great danger. The result of this fall in blood pressure has an immediate and disastrous effect upon the cerebral ganglia—and if the pressure remain too long reduced, irreparable damage takes place in these tissues. Moreover, a pressure below 30 millimeters of mercury in the coronary arteries precludes the possibility of cardiac action. That shock is entirely a question of vasomotor fatigue is not contended. The studies of Henderson are conclusive that there may be a condition of carbon dioxide exhaustion—apnea, oxygen starvation, and colloid change—which also may arise as a crisis incident to anesthesia or trauma. These may be designated as the chemic phenomena of shock, or acapnea. Between the theories of vasomotor exhaustion and acapnea the majority of all the cases of surgical shock fall.

If in the course of an operation those phenomena

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